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Critical Illness Polyneuropathy: Case Report and Update

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***Abstract* —** *Asthenia is a manifestation commonly found in critically ill patients hospitalized in an intensive care unit. In addition to the hard weaning from invasive mechanical ventilation, it is essential to recognize Critical Patient's Polyneuropathy (CPP). Case Report: JCD, 63 years old, white, single, obese (BMI> 40), type II diabetic controlled with diet and Metformin 2g / day, mild hypertension, with hyperuricemia (gout). Former smoker and social drinker. A clinic of severe abdominal pain, in bar type, appeared on the upper region of the abdomen on 07/07/2018, with a diagnostic hypothesis of urolithiasis and urinary infection. He was admitted on 10/07/2018 with an abdominal septic shock / cholecystitis. He remained on mechanical ventilation and use of amines for a long time, was tracheostomized and managed to decanulate. During this period, he developed ARDS lung, acute dialysis renal failure (recovered renal function), drug hepatitis and critical patient's polyneuropathy. Discharge from ICU on 28/09/2018. The examination reveals tetraparesis with a predominance of the distal, brachial and crural thirds; styloradial and flexor reflexes of the left hypoactive fingers, patellar hyporeflexia, abolished aquileus; superficial sensitivity: thermal and painful tactile hypoesthesia with paresthesia on the feet soles; profound sensitivity: proprioceptive hypoesthesia and hypopalesthesia in the distal third of the 4 limbs; in addition to flaccid paraparetic gait. Lumbar puncture, ENMG and biopsy of the sural nerve. In the CSF, there was an absence of leukocytes, normal glucose and 20mg / dL proteins.The ENMG showed axonal motor sensitive polyneuropathy, with decreased potential amplitudes. Discussion: Critical Patient's Polyneuropathy is a predominantly motor condition, however, related to sensitive, symmetrical and acute impairment associated with exaggerated systemic inflammatory response syndrome (SIRS), which*

mostly occurs in cases of sepsis. There is widespread asthenia and difficulty in weaning from invasive mechanical ventilation as the two peculiarities sine qua non for the identification of patients with CPP. The diagnosis of CPP is by exclusion. Electroneuromyography, in turn, is defined as the gold standard exam. Conclusion: Despite its occurrence, elements that are objectively related to its pathophysiology remain hidden. For this reason, the importance of further studies regarding the risk factors and diagnosis of CPP is reiterated in order to optimize the identification and the control of the severity of the affected patients.

I. INTRODUCTION

Asthenia is a manifestation commonly found in critical patients hospitalized in the intensive care unit (ICU), occurring in about 46% of patients hospitalized in the ICU [1,2] and who were exposed to the following risk factors: systemic inflammatory response syndrome/sepsis; multiple organ failure; hyperglycemia; dialysis; administration of vasoactive drugs, such as catecholamines; women; high mechanical ventilation time; use of corticosteroids; neuromuscular blockers. In addition to the hard weaning from invasive mechanical ventilation, it is essential to recognize Critical Patient's Polyneuropathy (CPP). [3]

There are reports of two comorbidities responsible for the generalized weakness that affect patient admitted to the ICU: Critical Patient's Polyneuropathy (critical illness polyneuropathy), [4] with acute polyneuropathy that most affects these patients, and critical myopathy (critical illness myopathy), which refers to muscle impairment primarily, with no nerve damage. [5]

Since the 1970s, neuromuscular function disorders have been recognized as the main cause of generalized asthenia, in addition to prolonged invasive mechanical ventilation (IMV), due to nerve and muscle damage in patients in the ICU. Such obtained muscle weakness is not only related to loss of muscle mass due to the long period of immobility or seriousness of the underlying pathology, but also to polytrauma, multiple organ dysfunction or severe infections, highlighting that the permanence, as well as the progress of illness resulting from hospitalization and the established treatment can provide the development of Critical Patient's Polyneuropathy. [6,7]

The term "Critical Patient's Polyneuropathy" has an axonal nature, mostly motor, symmetrical and acute. This expression was first described in 1984 by Bolton et al. in which they described a primarily motor condition, of an axonal, symmetrical and acute nature, in patients hospitalized in the ICU. Such patients had tetraparesis, abolished deep reflexes, as well as difficulty in removing IMV. The main cause related to the episode is the systemic inflammatory response syndrome (SIRS), mostly triggered by sepsis. [8] However, Critical Patient's Polyneuropathy is still a condition not completely clarified regarding its pathophysiology and etiopathogenesis. Thus, the aim of the present study is to expose a case of Critical Patient's Polyneuropathy, in order to draw attention to the diagnosis of this condition, which is not uncommon.

II. CASE REPORT

JCD, 63 years old, white, single, obese (BMI> 40), type II diabetic controlled with diet and Metformin 2g / day, mild hypertension, with hyperuricemia (gout). Former smoker and social drinker A clinic of severe abdominal pain, in bar type, appeared on the upper region of the abdomen on 07/07/2018, when he was urgently attended at Hospital Icaraí a diagnostic hypothesis of urolithiasis and urinary infection was made. He was medicated with oral antibiotic therapy, analgesics and oriented to outpatient follow-up. He evolved with persistent pain, abdominal stiffness, fever, vomiting and mental confusion. He was admitted on 10/07/2018 to the aforementioned hospital with abdominal septic shock / cholecystitis. He underwent laparoscopy with cholecystectomy on 11/07/2018. A post-surgical coma was induced, it remained so for 58 days. He developed a biliary fistula, requiring laparotomy with placement of drains. The septicemia persisted, a new abdominal approach was performed with washing of the cavity and collection of material for culture. He remained on mechanical ventilation and use of amines for a long time, was tracheostomized and managed to decanulate. During this period, he developed ARDS lung, acute dialysis renal failure (recovered renal function), drug hepatitis and critical patient's polyneuropathy. Discharge from ICU on 28/09/2018. Transferred to the Niterói Hospital Complex on 09/11/2018 where he used Meropenem and Polymyxin B for nine weeks. An echocardiogram showed aortic valve endocarditis. He was discharged from hospital on 03/05/2019 for home treatment (homecare). Hospitalized on 13/11/2019 for surgical exploration of saccular lesion in the left trochanteric region. There was dehiscence of the scar, he

remained hospitalized for 30 days. Presently, he remains in homecare, undergoes daily dressing on the remaining ulcer (sacral) and physical therapy for motor rehabilitation due to the developed neuropathy. He uses Pregabalin 300mg / day since the first hospitalization, Adera D3 7,000UI / alternate days and AAS 100mg / day. He wanders short distances with the aid of canadian crutches and needs assistance for daily activities. The examination reveals tetraparesis with a predominance of the distal brachial and crural thirds; styloradial and flexor reflexes of the left hypoactive fingers, patellar hyporeflexia, abolished aquileus; superficial sensitivity: thermal and painful tactile hypoesthesia with paresthesia on the feet soles; profound sensitivity: proprioceptive hypoesthesia and hypopalesthesia in the distal third of the 4 limbs; in addition to flaccid paraparetic gait. Lumbar puncture, ENMG and biopsy of the sural nerve. In the CSF, there was an absence of leukocytes, normal glucose and 20mg / dL proteins. ENMG showed axonal motor sensitive polyneuropathy, with decreased sensory and motor potential amplitudes in the fibular nerves, increased distal latencies and decreased nerve conduction speed in the tested groups of the lower limbs, in addition to positive waves and fibrillations in upper and lower limbs muscles. In the biopsy of the sural nerve, the semi-thin sections included in glutaraldehyde showed axon-myelinic neuropathy in activity without an inflammatory process.

Fig.1: Muscular atrophy with shedding of the feet (distal crural third)

III. DISCUSSION

Critical Patient's Polyneuropathy is a predominantly motor condition, however, related to sensitive, symmetrical and acute impairment associated with exaggerated systemic inflammatory response syndrome (SIRS), which mostly occurs in cases of sepsis, [9] during which changes occur in microcirculation, release of inflammatory cytokines and failure in the self-regulation of blood vessels responsible for innervation of peripheral nerves, succeeding in endoneural edema. Subsequently, there is hypoxia and consequent energy deficits that favor, the primary axonal degeneration of sensory and motor fibers, primarily distal, thanks to the encompassing of axonal transport totally dependent on energy, enabling cytokines and the tumor necrosis factor, to act negatively on the nerve peripheral. [6] CPP provides prolonged mechanical ventilatory support and difficulty in weaning, in addition to a long hospital stay. [10,11]

CPP is an acute axonal neuropathy that manifests itself as a consequence of increased survival in patients with multiple organ failure and sepsis. It presents a reversal of the condition, right after the control of the individual's critical condition. The disease manifests itself in a self-limited and monophasic form, with excellent recovery in patients with mild to moderate forms of the disease. [12]

There is widespread asthenia and difficulty in weaning from invasive mechanical ventilation as the two peculiarities sine qua non for the identification of patients with CPP. [13] Such characteristics happen in a synchronous manner, and are observed regardless of the reestablishment of the level of consciousness and clinical improvement. [14]

Critical Patient's Polyneuropathy has several variables with regard to severity and mainly affects the lower limbs, in which the distal region is the most severely affected. Mild sensory disturbances can occur. Occasionally, there are no dysautonomic manifestations. It starts in a subtle way, often only being recognized after adequate control of sepsis complications or multiple organ failure. [15]

A study by Garnacho-Montero et al. (2005), demonstrated that CPP has a strong impact on the time of mechanical ventilation, as well as typifying an isolated predictive factor of difficulty in weaning from mechanical ventilation, causing an average of 34 days of stay on IMV and a reintubation percentage of 41%. [16]

Another study with a prospective model carried out by De Letter et al. with 98 critically ill patients was evidenced that 33% of them evolved with neuromuscular disorders during their ICU stay. SRIS and the magnitude of the disease were identified as the only independent risk factors observed. Further research has shown that the use of corticosteroids stands out as another risk factor normally seen, being identified as the most relevant

predictive factor for the occurrence of muscle weakness in critically ill patients. [17]

Presently, the following are the diagnostic criteria for CPP: (1) immediate generalized asthenia for the establishment of critical illness; (2) diffuse weakness, affecting the proximal and distal muscles, symmetrical, flaccid and which normally preserves cranial nerves; (3) dependence on mechanical ventilatory support; (4) MRC score below 48 points, observed at different times, with an interval of 24 hours; (5) other causes of asthenia excluded. [18]

The diagnosis of CPP is by exclusion. It is essential to rule out metabolic, pharmacological and central causes (Central Nervous System) of asthenia, as well as spinal cord injury and intoxications. The gold standard diagnosis of CPP is performed by means of an electroneuromyography exam, which reveals the pattern of axonal polyneuropathy. Electroneuromyography, in turn, is defined as the gold standard exam. Its results reveal potentials of low amplitude motor and sensory actions; conduction velocity and moderately conserved distal latencies; fibrillations and positive waves may occur in needle stimulation. [19]

It is essential to rule out the likelihood of spinal cord injury in patients who develop muscle weakness after trauma; application of significant amounts of medication, leading to porphyria, in patients with acute intermittent porphyria; previous history of decompensated muscle, motoneuron or nerve diseases in patients in whom the reason for acute respiratory failure has not been elucidated clinically; patients with metastatic micro abscesses that can lead to peripheral nerve damage. Furthermore, it is important to emphasize that the reason for hospitalization is evident. [20]

There is no evidence of abnormalities in the cerebrospinal fluid (CSF) examination or it presents with slight changes. [21,22] Nerve biopsy shows primary axonal degeneration without evidence of an inflammatory process. [23] The disparity in certain conditions between pathological findings and the severity of clinical manifestations speaks in favor of axonopathy. It has a good recovery if the underlying disease is controlled. [24]

IV. CONCLUSION

Despite its occurrence, elements that would objectively be related to its pathophysiology, as well as the therapeutic ideal for the management of this condition, remain hidden. For this reason, the importance of further studies regarding the risk factors and diagnosis of CPP is reiterated in order to optimize the identification and control of diseases to affected patients.

REFERENCES

[1] Bretãs JRS, Oliveira JR, Yamaguti L. Reflexões de estudantes de enfermagem sobre morte e o morrer. Rev Esc Enferm USP. 2006;40(4):477-83.

[2] Pitta A. Hospital: dor e morte como ofício. Hucitec. 1994: 4.

[3] Viana A, Picelli H. O estudante, o médico e o professor de medicina perante à morte e o paciente terminal. Rev Bras Educ Med. 1998;44(1):21-7.

[4] Kovács MJ. Educação à morte: desafio na formação de profissionais de saúde e educação. Casa do Psicólogo. 2003: 117.

[5] Sousa DM, Soares EO, Costa KMS, Pacífico ALC, Parente ACM. Vivência da enfermeira no processo de morte e morrer dos pacientes oncológicos. Texto & Contexto Enfermagem. 2009; 18(1):41-7.

[6] Canineu RFB et al. Polineuropatia no paciente crítico: Um diagnóstico comum em medicina intensiva?, Revista Brasileira de Terapia Intensiva. 2006; 18 (3):307-310.

[7] Schaaf m van der, Beelen a, Vos r. de. Functional outcome in patients with critical illness polyneuropathy, Disability and Rehabilitation, Amsterdam 2004; 26 (20): 1189-1197.

[8] Druschky A, Herkert M, Radespiel-Troger M, et al - Critical illness poly Critical illness polyneuropaty: clinical findings and cell culture assay of neurotoxicity assessed by a prospective study. Intensive Care Med, 2001;27:686-693.

[9] Alvim LBAM, Freitas MRG, Nascimento OJM, et al - Polineuropatia do paciente crítico: registro de caso. Arq. Neuro Psiquiatr. 1999, 57 (2A): p.317-322. Disponível em: . ISSN 0004-282X.

[10] De Jonghe B, Sharshar T, Lefaucheur J, et al - Paresis acquired in the intensive care unit: a prospective multicentre study. JAMA, 2002; 288:2859- 2867.

[11] Naeem A, O'Brien J, Hoffmann S, Philips G, Garland A, Finley JC, Almoosa K, et al. Acquired weakness, handgrip strength, and mortality in critically ill patients. Am J Respir Crit Care Med. 2008, 1;178(3):261-8. http://dx.doi.org/10.1164/rccm.200712-1829OC

[12] Hund E - Neurological complications of sepsis: critical illness polyneuropathy and myopathy. J Neurol, 2001;248:929-934

[13] Silva GSN, Ayres JRCM. O encontro com a morte: à procura do mestre Quíron na formação médica. Rev Bras Educ Med. 2010, 34(4):487-96

[14] Lock M. Twice dead: organs transplants and the reinvention of death. Berkeley: University of California Press; 2001: 233.

[15] Zochodne DW, Bolton CF, Wells GA et al - Critical illness polyneuropathy. A complication of sepsis and multiple organ failure. Brain, 1987;110:819- 841.

[16] Garnacho-Montero J, Amaya Villar R, Garcia-Garmendia JL, Madrazo-Osuna J, Ortiz-Leyba C. Effect of critical illness polyneuropathy on the withdrawl from mechanical

ventilation and the length of stay in septic patients. Crit Care Med. 2005;33:349-53

[17] De Letter MA, Schmitz PI, Visser LH, Verheul FA, Schellens RL, Op de Coul DA, et al. Risk factors for the development of polyneuropathy and myopathy in critical ill patients. Crit Care Med. 1998; 24(8): 801-807

[18] Stevens RD, Marshall SA, Cornblath DR, Hoke A, Needham DM, de Jonghe B, et al. A framework for diagnosing andclassifying intensive care unitacquired weakness. Crit Care Med. 2009, 37(10 Suppl):S299-308. http://dx.doi.org/10.1097/CCM.0b013e3181b6ef67

[19] Dhand U. Clinical approach to the weak patient in the Intensive Care Unit. Respir Care 2006; 51 (9): 1024-1041

[20] Rich MM, Teener JW, Raps EC et al - Muscle is electrically inexcitable in acute quadriplegic myopathy. Neurology. 1996;46:731-736.

[21] Hund EF, Fogel W, Krieger D, DeGeorgia M, Hacke W. Critical illness polyneuropathy:clinical findings and outcomes of a frequent cause of neuromuscular weaning failure. Crit Care Med 1996;24:1328-1333.

[22] Op-de-Coul AA, Verheul GA, Leyten AC, Schellens RL, Teepen JL. Critical illness polyneuromyopathy after artificial respiration. Clin Neurol Neurosurg. 1991;93:27-33.

[23] Leijsten FS, De Weerd AW. Critical illness polyneuropathy: a review of the literature, definition and pathophysiology. Clin Neurol Neurosurg. 1994;96:10-19.

[24] Bolton CF. Eletrophysiologic studies of critically ill patients. Muscle Nerve. 1987;10:129-135.